A DISSERTATION ON

CLINICAL OUTCOME OF GASTRIC ULCER PERFORATION - A PROSPECTIVE STUDY

Dissertation submitted to

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

In partial fulfillment of the requirement for the degree of

M.S. DEGREE IN GENERAL SURGERY

BRANCH – I

MADRAS MEDICAL COLLEGE

RAGIV GANDHI GOVERNMENT GENERAL HOSPITAL

CHENNAI – 600 003.

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CERTIFICATE

This is to certify that the dissertation titled "**A STUDY ON CLINICAL OUTCOME OF GASTRIC ULCER PERFORATION**" is the original work done by **Dr.S.Venkatesan**, post graduate in M.S., General surgery at the department of general surgery, madras medical college, Chennai 600 003 to be submitted to the Tamilnadu Dr.M.G.R Medical university, Chennai 600 032, towards the partial fulfillment of the requirement for the award of M.S.,degree in General Surgery during the academic period from may 2010 – April 2013.

Dr.V.KANAGASABAI M.D.,
Dean
Madras Medical college,
Chennai – 600 003

PROF S.DEIVANAYAGAM M.S.,
Professor & Head of department,
Department of General surgery,
Chennai – 600 003

PROF P.RAGUMANI M.S.,
Professor,
Department of General Surgery,
Madras medical college,
Chennai - 600 003.

DECLARATION

I solemnly declare that the dissertation titled **" CLINICAL OUTCOME OF GASTRIC ULCER PERFORATION – A PROSPECTIVE STUDY "** was done by me at Rajiv Gandhi Government General Hospital, Chennai during the period of August 2012 to December 2012 under the guidance and supervision of

Prof P.Ragumani M.S.,

The dissertation is submitted to the Tamil Nadu Dr.M.G.R Medical University, Chennai towards the partial fulfillment of the requirement for the award of **MS Degree in General Surgery Branch –I**

Dr.S.Venkatesan

Place :

Date :

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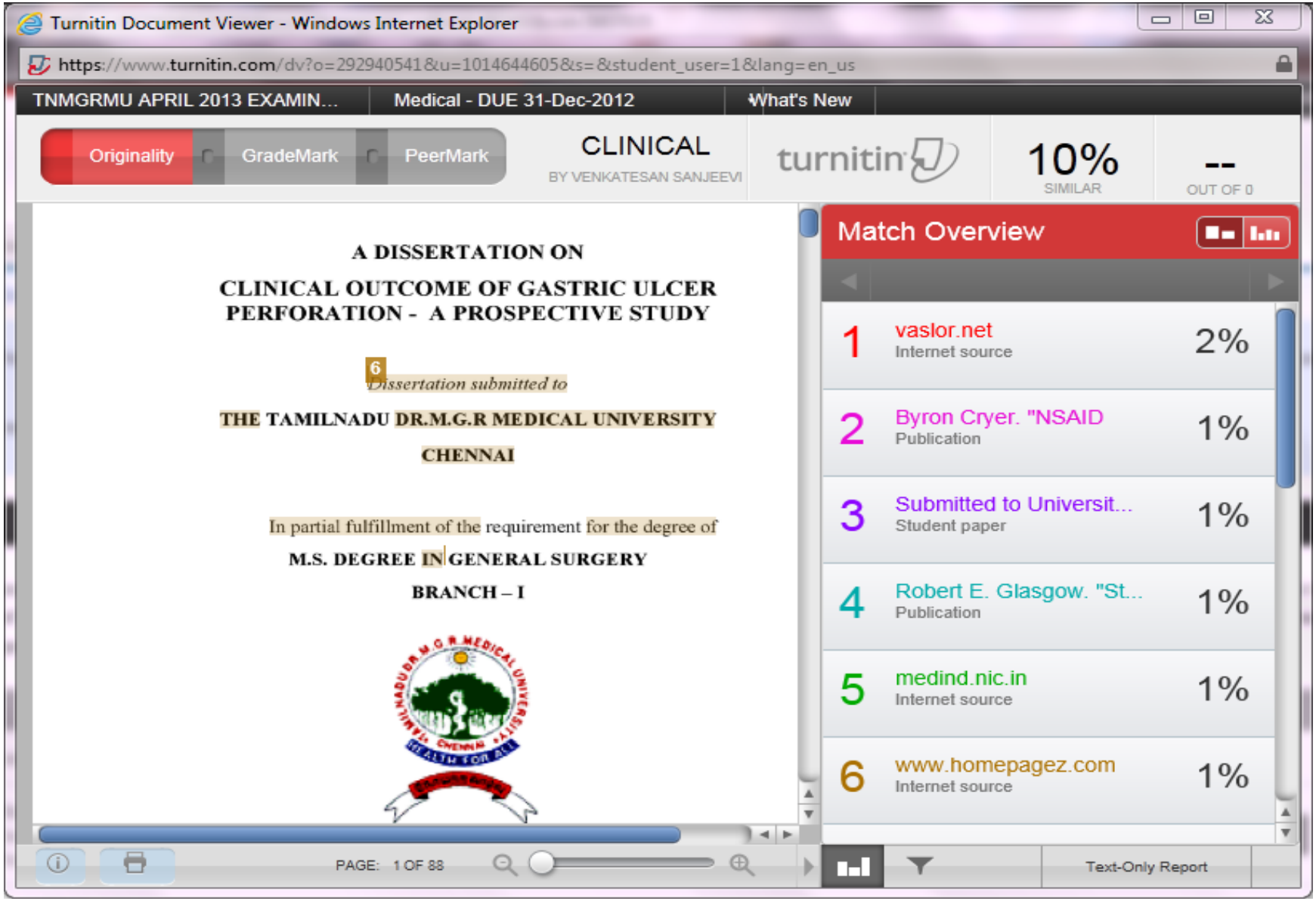
Dr.S.VENKATESAN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ABBREVIATION

SC WITH OP	Simple closure with omental patch
POD	Post operative day
GA	General anesthesia
DM	Diabetes mellitus
IHD	Ischemic heart disease
CRF	Chronic renal failure
COPD	Chronic obstructive pulmonary disease
COX	Cyclo oxygenase
EGF	Epidermal growth factor
FGF	Fibroblast growth factor
HCO3-	Bicarbonate
H+	Hydrogen ion
Cl-	Chloride ion
APD	Acid peptic disease
GIT	Gastro intestinal tract
NSAID	Non steroidal anti inflammatory drug
H.PYLORI	Helicon bacter pylori

CONTENT

HISTORICAL REVIEW

Acute perforation of peptic ulcer is relatively a common complication. It was rarely reported 100 years ago. There is progressively an increase in its incidence during the last few decades in India.. In the year 1944, Illingworth has shown from his 20 years study from 1924 to 1944, a fivefold increase in the incidence of gastrointestinal perforations.

Warren Cole assessed the occurrence of perforations in chronic duodenal ulcer and in chronic gastric ulcer was 20.5%.

Rawlison was credited with the first published report in 1727 of a perforated gastric ulcer. The first published report of a perforated duodenal ulcer was by Hambergeiri in 1946.

Heusner was the first to close a perforated duodenal ulcer successfully. Simple closure of a perforated ulcer was done in 1892 by Kriege.

Cellen Jones in 1929 described the most widely used method of closing aperforation with a live omental patch, often wrongly credited to Roscoe Graham.

Moore and colleagues in 1950 found that recurrence of ulcer symptoms after repair of a perforation carried a bad prognosis in their 10 year follow up analysis of 1000 ulcer patients.Collier and Pain in 1985 reported that 45% of the patients aged 15 years or more presenting with perforated ulcer had consumed NSAIDs.

Watkini et al. in 1984 found that 25% of the patients in the Oxford area were consuming NSAIDs, and 4.8% were taking steroids at the time of perforation.

Hamilton and Harbrecht in 1967 and Khan and Ralston in 1970 reported that operative mortality of truncal vagotomy with PGJ is about 1%. Jordan, De Bakey and Duncan in 1974 reported 535 emergency partial gastrectomies with an operative morality of 2.2%.

J S Pierandozzi, B B Hin Shaw and O E Stafford in 1960 treated perforated peptic ulcer by vagotomy and pyloroplasty.

Laparoscopic treatment was reported in the year 1990. Mouret et al. found that laparoscopic management is good because of avoiding large incision, decrease in the wound infection and good peritoneal lavage. He treated 4 out of 5 patients successfully.

In 1997 John Wayman and Simon A Raimes found that simple closure treatement is safe and effective in long term, when combined with H.pylori eradication and pharmacological suppression.

INTRODUCTION

Gastric ulcer perforation is one of the acute abdominal emergencies in the surgical field.

The incidence of peptic ulcer disease has been declining for the past 25 years and the need for the elective ulcer surgery is on decline, neither the incidence nor the need for surgery for the emergent complications of the ulcer(perforation, bleeding and obstruction) have changed significantly during the past 20 –25 years.

This study deals with one of the complications of peptic ulcer, namely gastric ulcer perforation, trends in age distribution of occurrence, risk factors, outcome of operative treatment and factors influencing the prognosis of the disease.

Gastric ulcer in the lesser curvature near the antrum perforates. Amount of gas escaped is more than the perforated duodenal ulcer. Malignancy should always be suspected and so biopsy from the edge of the ulcer is a must.

Mortality in gastric ulcer perforation is high, commonly they are pre pyloric in position. Primary closure with an edge biopsy is commonly

used. Distal gastrectomy including ulcer area is better option if patients general condition is favourable.

Posterior gastric ulcer perforation is often difficult to diagnose both clinically and radiologically.

Detailed history, good physical examnation and good clinical acumen plays a major role in diagnosing this acute abdominal emergency.

There is multiple factors influencing the progress of the disease and its prognosis which be discussed in detail in this study.

SURGICAL ANATOMY OF THE STOMACH

Stomach is part of the embryonic foregut. It is an ovoid musculomembranous digestive pouch below the esophagus.

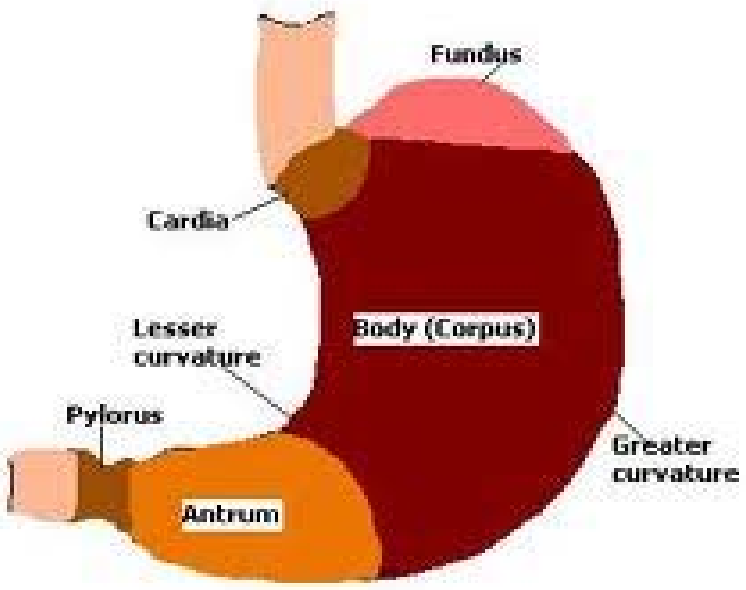
The end whichconnects with esophagus is the cardiac end the end that in continuous with the duodenum is the pyloric end.

The stomach measures about 25 cm in length and 10 cm in diameter. It has a capacity of 0.9 to 1.4 litres.

The wall of the stomach consists of serosa, muscularis propria which inturn consists of longitudinal, circular and oblique firbes, submucosa, muscularis mucosa and mucosa from outwards.

The secretions of the stomach is gastric juice containing pepsin, mucus and Hcl.

STOMACH



SURGICAL ANATOMY OF PERITONEAL CAVITY

The peritoneal cavity is lined with a single layer of mesothelial cells.The parietal peritoneum covers the abdominal cavity (i.e., abdominal wall, diaphragm, pelvis), the visceral peritoneum covers all the

intra abdominal viscerae, forming a cavity that is completely enclosed except at the open ends of the fallopian tubes.

A small amount of fluid sufficient to allow movements of organs is usually present in the peritoneal cavity. The fluid is normally serous (protein content <30 g/l, < 300 WBCs/µl). In the presence of infection, the amount of this fluid increases, the protein contents climbs to more than 30 g/l and the WBC count increases to more than 500 WBCS/µl, in other words, the fluid becomes an exudate.

The transverse colon and the drape of greater omentum divide the abdomen horizontally into supracolic and infracolic compartments. Therefore the symptoms and signs of peritonitis may be localized to upper or lower halves of the abdomen for sometime.

The forward convexity of the lumbar spine provides two marked lateral gutters and only a shallow anterior communication between them across the midline. Consequently, liquid spreads by movement largely around the periphery of the abdomen and not a great deal across the midline, hence the initial laterality of many peritoneal processes.

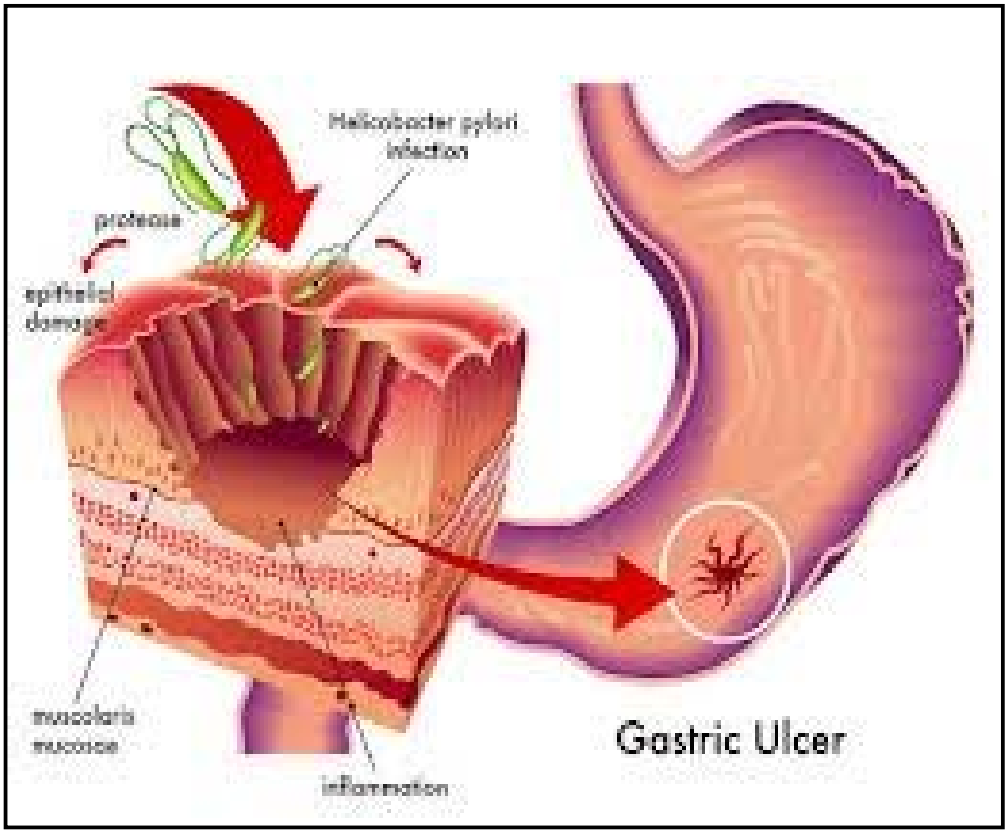
The right subhepatic space (Morison's pouch) is open only to the right, where it communicates with the right paracolic gutter. Liquid from

perforated duodenal ulcer or seepage from the gallbladder region passes to the right and then both upwards to reach the right subphrenic space and downwards to the right iliac fossa. Thus, on one hand, there is subphrenic abscess and shoulder tip pain and on the other hand, the occasional diagnostic confusion between appendicitis and either perforated peptic-ulcer or acute biliary tract conditions.

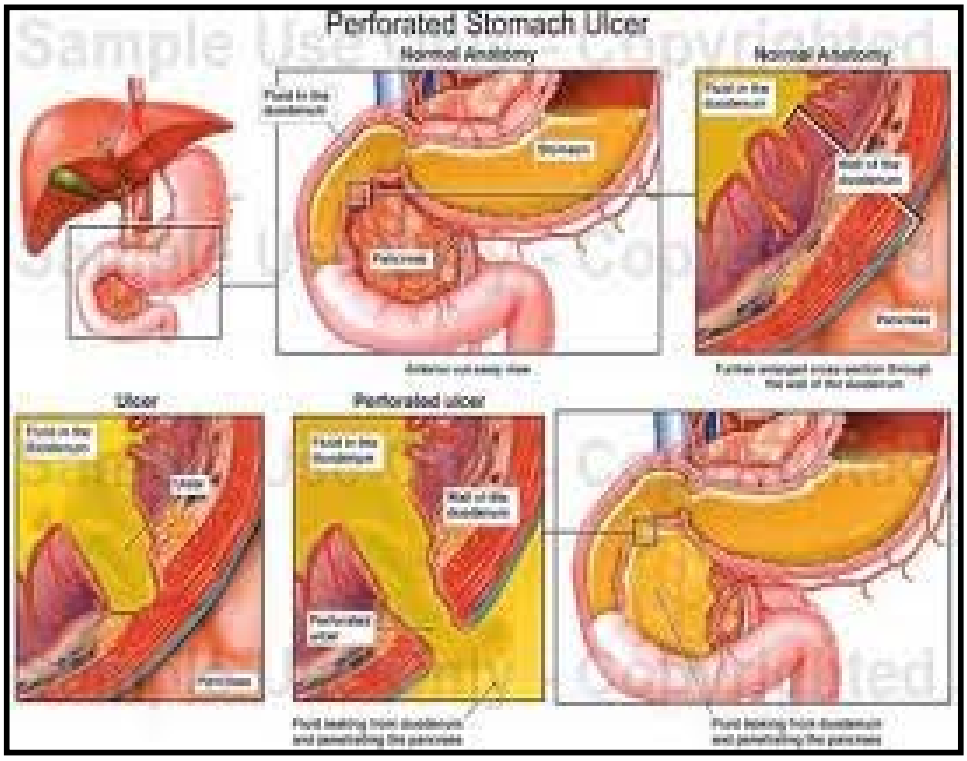
Paracolic effusions reach the general peritoneal cavity across the sigmoid flexure. Pelvic effusions pass up both the paracolic gutters and there after to the subphrenic spaces and to the general peritoneal cavity.

A left sided origin above the transverse colon results in left paracolic and left subphrenic spread.

GASTRIC ULCER



GASTRIC ULCER PERFORATION



AIM OF THE STUDY

The aim of the study are

- To study the age group, sex, causes, socioeconomic status, personal habits and
- sensitivity of investigational modalities in diagnosis of gastric ulcer perforation.
- To study the risk factors.
- To study the prognostic factors influencing the disease process.
- To study the outcome of operative treatment – morbidity and mortality.
- Biopsy study from the edge of the ulcers and incidence of malignant ulcer perforation.

REVIEW OF LITERATURE

Gastric ulcer perforation is an acute abdominal emergency. Spillage of gastric contents into the peritoneal cavity resulting in peritonitis, electrolyte and fluid imbalance, circulatory insufficiency, septicaemia and finally death.

The peritoneal cavity is lined by mesothelial cells- single layer. Parietal peritoneum covers the abdominal cavity. Visceral peritoneum covers all intra abdominal viscerae. peritoneal fluid is serous, in presence of infection the amount of fluid increases and becomes exudates.

The transverse colon and greater omentum divides the abdomen into supracolic and infra colic compartments, so symptoms and signs of peritonitis localized to upper or lower halves of abdomen.

Morison's pouch is open only to the right and communicates with right paracolic gutter.

Incidence of perforated peptic ulcer is approximately 8 – 10 cases per 10000000 per year. There has been a significant change in the epidemiology of perforated peptic ulcer over the last 2 decades.

Previously more common in the middle aged between 30-50 years. Now increasing use of NSAID have resulted in an increased incidence in <30 age. Perforation is present in about 8% of patients hospitalized for peptic ulcer disease and is the first manifestation of the disease an about 2-3% of patients with gastric ulcer.

It is estimated that, after the diagnosis of gastric ulcer, 0.4% of patients perforate annually in the first 10 years.

Whether eradication of H.pylori will reduce this incidence is as yet unknown. Usually anteriorly placed gastric ulcer perforates, and the aphorism that anterior ulcer perforate, posterior ulcer bleed is as relevant today as ever.

ACUTE PEPTIC ULCERS:

- NSAIDS
- Stress
- Sepsis
- Cushing's ulcer
- Curling's ulcer
- Steroids.

CHRONIC PEPTIC ULCERS:

Gastric ulcer patients secrete either low normal or below normal amount of acid. Only 5% of patients may demonstrate acid hypersecretion.

- ❖ diminished mucosal resistance
- ❖ pyloro duodenal reflux
- ❖ deficient mucosal barrier
- ❖ mucosal trauma
- ❖ local ischemia
- ❖ antral stasis
- ❖ NSAIDS
- ❖ helico bacter pylori
- ❖ malignancy

Gastric ulcers approximately 80-85% found on or near lesser curvature, the great majority of these being closure to the incisura angularis. Only 10% of chronic gastric ulcers are found in the antrum, 2% in the pyloric canal, 3% in the cardia and 5% in the fundus and body of the stomach.

Few simple ulcers undergo malignant degeneration. True incidence of carcinoma developing in a benign ulcer is not very definite. But it may not be more than 1%. Giant ulcers (more than 2.5cms in diameter) may be considered suspicious, though there are also rarely carcinomatous.

Acute gastric ulcers are frequently multiple in 75% of cases. These can occur in any part of the stomach.

These ulcers are oval or circular in shape and usually vary in size from 1 to 2 mm in diameter to about 1 cm in diameter.

The main features of these ulcers are that they are shallow, punched out and usually do not invade muscular coat.

Peptic ulceration results when the caustic effects of acid and pepsin in the gastrointestinal lumen overwhelm all three components of epithelial defense.

The mechanisms that enable the mucosa to resist acid peptic attack can be divided into three major components – pre epithelial, epithelial and post epithelial defence mechanisms.

1.PRE EPITHELIAL DEFENCE MECHANISM :

The pre-epithelial defense mechanism are features that impede contact between epithelial cells and noxious agents in gastrointestinal lumen

Gastric epithelial cells are normally protected from acid –peptic attack by the coat of mucus and by a layer of unstirred water which is rich in HCO3.

Both are secreted into lumen by gastric epithelial cells. Bicarbonate from the blood also enters the unstirred water through the process of paracellular diffusion.

Within the mucus layer glycoproteins form a physical barrier and HCO3 ions that accompany the glycoproteins can neutralizes the acid, mucus also contains substantial quantities of surface active phospholipids which are secreted by epithelial cells.

These phospholipids may guard the mucosa by forming a hydrophobic layer that repels acid at the luminal surface of the mucus gel.

As a result of pre epithelial defense mechanisms, the pH on the surface of the gastric epithelial cell normally can be maintained in the neutral range, even when the pH in the lumen falls below 2.6.

Finally acid peptic injury to the gastric mucosa results in outpouring of mucus, fibrin and cellular debris forms a protective cap and impedes further contact with acid. So any abnormalities in the epithelial mechanisms will contribute to peptic ulcer disease.

For example helico bacter pylori can be associated with abnormalities in the gastrointestinal mucus secretion and in duodenal bicarbonate secretion that predispose to peptic ulceration.

2.EPITHELIAL DEFENCE MECHANISM :

When acid and pepsin break the pre-epithelial defenses, epithelial mechanisms can prevent or minimize acid – peptic injury.

The apical cell membranes and tight junctional complexes between the surface cells are barriers that limit the diffusion of hydrogen ions into the mucosa.

Exposure to more concentrated acid induces injury that allows hydrogen ions to leak through this paracellular pathway. Excess H+ ions

that enter the epithelial can be removed by ion pumps that include a Na+/H+ exchanger and a Cl-/HCO3- exchanger.

Gastric epithelial cells also have a Na/HCO3 cotranspoter that helps to regulate intracellular pH.

When these defense mechanisms are over whelmed and cells succumb to acid –peptic injury, superficial mucosal defects can be sealed quickly by a process 'rapid restitution' in which healthy cells in the mucus neck region of the gland migrate along the basement membrane to close the mucosal gap. This process is regulated by EGF and FGF.

Rapid restitution merely involves cell migration, not cell division and the wandering cells can seal only minor mucosal defects.

Healing of large ulcers is effected through regeneration, a process in which new cells are created by cell division. Regeneration also regulated by growth factors.

3.POST EPITHELIAL DEFENCE MECHANISM :

Mucosal blood flow comprises the post epithelial defense mechanism. Bloodflow gives energy and substrate required for both maintaining epithelial cell integrity, mucus production and HCO3

secretion. And it also removes acid that diffuses through an injured mucosa.

During gastric acid secretion, bicarbonate transported across the the parietal cell membrane produces an alkaline tide in the submucosa, protect against acid –peptic injury during acid secretion by the stomach.

Peptic ulceration results when caustic effect of acid and pepsin in the gastrointestinal lumen overwhelm all three components of epithelial defence.

NSAID and peptic ulcer:

The majority of peptic ulcerations that are not associated with H.pylori infection are associated with NSAID ingestion.

NSAID induced ulcers can be symptomatic and complicated by GI bleeding , perforation and /or obstruction.

1% to 5% of patients receiving NSAIDs for more than one year will experience serious GI complications.superficial gastric lesions such as petechiae and erosions are found in approximately 60% of individuals who chronically consume NSAIDs, but these lesions appear to have little clinical importance.

Asymptamatic ulcerations can be documented endoscopically in 15% to 40% of patients on chronic NSAID therapy.

NSAID induced injury divided into two categories

1.those dependent on inhibition of the enzyme COX

2.those independent of enzyme COX inhibition

Topical NSAIDs are likely the major mechanism responsible for the acute hemorrhages and erosions observed acutely after NSAID challenge.

NSAID ingestion causes denudation of surface epithelial cells and increased mucosal permeability occur.

Most NSAIDs are weak organic acids that, in acidic gastric juice, are un-ionized and thus freely lipid soluble.

The lipid soluble, un-ionizes NSAIDs diffuse across the gastric mucosal epithelial cell membranes into the cytoplasm, where they ionize at neutral pH and thus become trapped with in the cells, causing uncoupling of oxidative phosphorylation, resulting in decreased mitochondrial energy production and increases in cellular permeability.

Another topical mechanism of NSAID injury is an attenuation of the phospholipid content and surface hydrophobicity of the gastric mucus layer.

Some NSAID metabolites excreted in bile causes topical injury to gastro intestinal mucosa.

Previous history of peptic ulcer disease is the most important risk factor for NSAID induced complication that increases the risk by 2 to 4 fold.

Advanced age is also a substantial risk factor. Although there does not appear to be a threshold age at which risk dramatically increases, the relative risk increases linearly at the rate of approximately 5% per year of advanced age.

Data on the role that duration of NSAID exposure has in the risk for gastrointestinal events have been conflicting. NSAID associated gastro intestinal complications is highest within the first 30 days of NSAID use.

If the dose of NSAID use is high the risk of ulcer complications also high. Other risk factors are concomintant use of flucocorticods or

anticoagulants and comorbid conditions like heart disese or rheumatoid arthritis.

NSAID use and helicon bacter pylori infection generally have been regarded as independent risk factors for peptic ulcer disease.

However evidence is accumulating that helicon bacter pylori infection and NSAID use may be more than just additive risk factors for ulcer disease.

NSAID users infected with H.pylori have two fold increased risk for developing bleeding peptic ulcer complications.

Cigarette Smoking

It is a risk factor for peptic ulcer disease and its complications. It affect the healing of peptic ulcerations and in the absence of treatment with H.pylori may predispose to relapse.

Smokers have low concentration of prostaglandins in their gastric mucosae, and smoking inhibit acid stimulated mucosal bicarbonate secretion.

Alcohol intake

A common misconception among clinicians is that alcohol ingestion is a strong risk factor for peptic ulcer disease. In fact few published data support this notion.

The prevalence of peptic ulcer disease appears to be increased for patients with alcoholic cirrhosis but not much association has been established for drinkers without cirrhosis. Indeed, one retrospective study suggested that modest alcohol consumption might even protect against peptic ulceration.

Diet

Usually ulcer patients complaints of dyspepsia associated with ingestion of foods like spicy foods. But the evidence that foods cause ulceration is virtually non existent.

Tea, coffee and colas are potent acid secretagogues. Both caffeinated and decaffeinated appear to be equal in their ability to stimulate gastric acid secretion.

MODIFIED JOHNSON'S CLASSIFICATION OF PEPTIC ULCER

- **TYPE 1** : Ulcer in body of the stomach along the lesser curvature at incisura sngularis.

- **TYPE 2**: Ulcer in both body of the stomach and duodenum associated with acid yyper secretion.

- **TYPE 3**: Ulcer in prepyloric channel within 3 cm of pylorus associated with acid hypersecretion.

- **TYPE 4**: Ulcer in proximal gastro oesophageal region.

- **TYPE 5**: Ulcer throught the stomach associated with NSAID use.

DISEASES ASSOCIATED WITH PEPTIC ULCER

A number of chronic illness associated with peptic ulcer disease eg. COPD as cigatette smoking may underlie both conditions.

Patients with cirrhosis appear to have an increased risk of developing peptic ulcerations and its serious complications.

Other diseases include

- cushing's disease,
- chronic renal failure
- hyperparathyroidism and
- coronary heart disease.

EMOTIONAL STRESS

Emotional stress alone does not appear to be sufficient to cause ulcers in most patient because eradication of H.pylori and elimination of NSAIDs generally prevents ulcer recurrence recurrence irrespective of emotional factors. Some modern studies still suggest that stress contributes to peptic ulcer disease.

Further more, it is not known why only a minority of individuals who take NSAIDs or who are infected with helicobacter pylori develop

peptic ulcers, and emotional stress and/or a genetic predisposition may well be risk factor is in these susceptible subjects.

GENETICS

A number of observations have suggested that genetic factors predispose to the development of ulcer disease.

Some of the familial clustering of ulcer disease is the result of a high rate of a H.pylori infection in family members.

An elevated level of serum pepsinogen-I level initially thought to be genetic marker for ulcer disease, also appears to be a reversible consequence of H.pylori infection.

The association of certain blood group antigens with peptic ulcer disease, these antigen may affect an individuals susceptibility to H.pylori infection. Eg lewis blood group antigen.

PATHOGENESIS OF GASTRIC ULCER PERFORATION

Perforation of an ulcer due to sudden sloughing of base of ulcer due to impairment of blood supply, which leads to gastric contents leaking into peritoneal cavity, initiating an acute diffuse peritonitis.

Although the initial peritonitis following perforation of peptic ulcer is a chemical one, there is mainly concurrent bacterial contamination which can aggravate the inflammatory process and progress to the development of intra abdominal abscesses over the ensuring days or weeks.

The important complications of acute peritonitis are paralytic ileus and toxaemia.

Absorption of bacterial endotoxins causes endotoxaemia that results in septicaemia.the combination of endotoxic shock and fluid,electrolyte imbalance causes high fatality rate if untreated.

BACTERIOLOGY

The most common organism found in the peritoneal fluid are

- E.Coli,
- Staphylococci,
- Streptococci and
- Anerobic organism.

H.PYLORI AND PEPTIC ULCER DISEASE

Recurrent peptic ulcer perforation more common with H.Pylori infection.

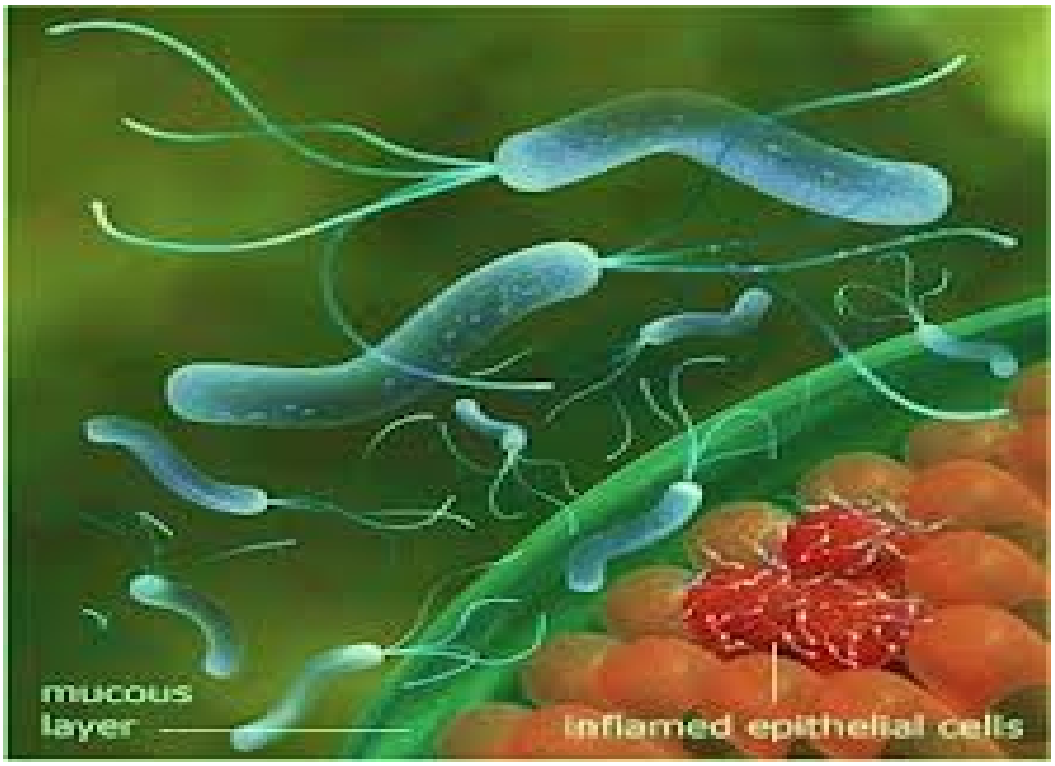
It is a

- microaerophilic,
- curved,
- Gram negative
- urease producing organism

H.pylori is able to colonize to surface of the mucosa, deep into the overlying mucus layer.

In 70% of the gastric ulcer patients infected with H.pylori. so antibiotic therapy for H.pylori will heals the ulcers and prevents their recurrence.

HELICO BACTER PYLORI



The mucosal barrier is breaked by the production of an endopeptidase which is a powerful mucolytic. Large amount of ammoniaproduced leads to alteration in epithelial surface, pHalteration in mucosal charge gradient,cellular permeability and NaKATPase activity leadind to back diffusion of hydrogen ion

Diagnosis by

- chromo endoscopy and
- breath urease test,

microscopically by

- Giemsa and
- warthin starry silver stain.

CLINICAL FEATURES

Perforations are classified into

- Acute massive perforation
- Slow perforation
- Sub acute perforation
- Chronic perforation

Clinical course of massive perforation divided into 3 stages.

1.First stage – Peritonism

It is due to irritation of peritoneum caused by perforation or inflamed viscus near about the pain is severe and is made worse by breathing and movement.

It is first experienced at the site of perforation and gradually spreads all over the abdomen. The patient may or maynot vomit.

On examination the patient usually lies still. There is little change in the pulse and temperature. Tenderness, rigidity and muscle guard are constantly present over the site of lesion.

It must be remembered in this context, that in case of pelvic peritonitis or peritonitis of lesser sac there is hardly any tenderness or rigidity of the anterior abdominal wall.

Rectal and vaginal examination along with palpation of the flanks and highly important. Infrequent bowel sounds will be heard as the paralytic ileus has not set in.

Diagnosis should be made at this stage as if the condition is allowed to continue the chance of survival of the patient will be minimized.

Two features are important for diagnosis at this stage viz onset of pain with dramatic suddenness and presence of muscle guard and rebound tenderness.

2. Second stage - Stage of Reaction.

At this stage the irritant fluid becomes diluted with peritoneal exudate. The intensity of the symptoms dwindles although the fire is still burning under the ashes.

The patient feels comfortable and nothing is more deplorable than the attending doctor sharing the patients comfort.

On examination muscle rigidity continues to be present though it may be softer the other two new features at this stage are obliteration of the liver dullness and appearance of shifting dullness. Paralytic sets in with silent abdomen on auscultation.

Rectal examination will still elicit tenderness in case of pelvic peritonitis in recto vesical or recto uterine pouch. At this stage straight X ray in sitting posture will reveal gas under diaphragm in 70% of cases.

3. Third stage - Stage of diffuse peritonitis.

At this stage patient has gone a step further towards the grave. The pinched and anxious face, shrunken eyes and hollow cheeks- the so called facies hippocratica is quite characteristic of this condition.

On examination the pulse rate rises and becomes low in volume. There is persistant vomiting. The abdomen becomes silent and increasingly distended. There is board like rigidity of the abdomen. The patient finally collapses into unconsciousness.

Slow perforation

Pain may be less severe, less generalized with definitive tenderness but equivocal guarding and rigidity where the perforation is. Bowel sound frequently persistant.

A small amount of fluid may track down the right para colic gutter, producing pain and tenderness in right iliac fossa, simulating appendicitis.

Subacute perforation

Ulcer may perforate and sealed rapidly, before contamination of general peritoneal cavity. Guarding and rigidity in epigastrium and right hypochondrium. Abdomen will be soft. This type of presentation is known as formed fruste.

Chronic perforation

When full thickness hole is created by an ulcer but free spillage is prevented by contiguous organ such as colon, greater omentum creating a walled of area. Signs and symptoms are absent.

Perforation and haemorrhage

The combination of haemorrhage and perforation occurs in 3 ways.

- Concomitant haemorrhage and perforation
- Perforation during medical treatment of haemorrhage
- Haemorrhage after a recently sutured perforation.

INVESIGATION

1. Complete blood count
2. Renal function test
3. Liver function test.
4. Serum electrolytes
5. Serum amylase
6. Pulmonary function test
7. Arterial blood gas analysis.
8. BT,CT
9. Blood culture.
10. Peritoneal fluid culture.

Leukocytosis with a left shift is usually present, but may be absent in the immunosuppressed or elderly patient.

Serum amylase is most commonly normal, but elevated levels less than three times normal are occasionally encountered.

Liver function tests are usually normal,unless the presentation is delayed, serum electrolytes and renal function are normal.

RADIOLOGICAL INVESTIGATION

X – RAY

A chest radiograph and supine and left decubitus abdominal films should be Taken.

Free air under diaphragm is seen in 70-80% of cases. The absence of free air does not exclude the diagnosis.

Causes of pseudopneumo peritoneum

1. Sub diaphragmatic fat
2. Omental fat
3. Subphrenic abscess

4. Sub pulmonary pneumothorax

5. Intra mural gas in pneumatosis intestinalis

6. Chiliaditi syndrome

7. Curvilinear pulmonary collapse

CONTRAST RADIOGRAPHY

Gastrograffin is used in doubtful cases to differentiate sealed from unsealed Perforation.

USG ABDOMEN/CECT ABDOMEN

It detect free intraperitoneal air and fluid in peritoneal cavity.

DIFFERENTIAL DIAGNOSIS

It can be

- Intra abdominal,
- Intra thoracic and
- Metabolic conditions.

I.Intra abdominal

- Acute appendicitis
- Acute cholecystitis
- Acute pancreatitis
- Acute intestinal obstruction
- Mesenteric thrombosis
- Traumatic perforation
- Ruptured ectopic

II. Intra thoracic causes

- Pleurisy
- Pneumonia
- Acute myocardial infarction
- Spontaneous pneumothorax
- Emetic rupture of oesophagus

III. Metabolic

- Acute porphyria
- Diabetes
- Uremia

- Multiple sclerosis
- Neurosyphilis

MANAGEMENT

INITIAL MANAGEMENT

- Insertion of naso gastric tube
- Intra venous fluids
- Bladder catheter to monitor urine output
- Broad spectrum intravenous antibiotics, central hemodynamic monitoring is indicated in unstable patients.

The goal of management should be promptly reestablish intravascular fluid volume in order to decrease the risk of anesthesia induction.

Prolonged efforts to establish a diagnosis and resuscitate these patients are counterproductive, as early operation is warranted.

Combination therapy with ampicillin or cephalosporin, gentamycin and metronidazole is commonly used.

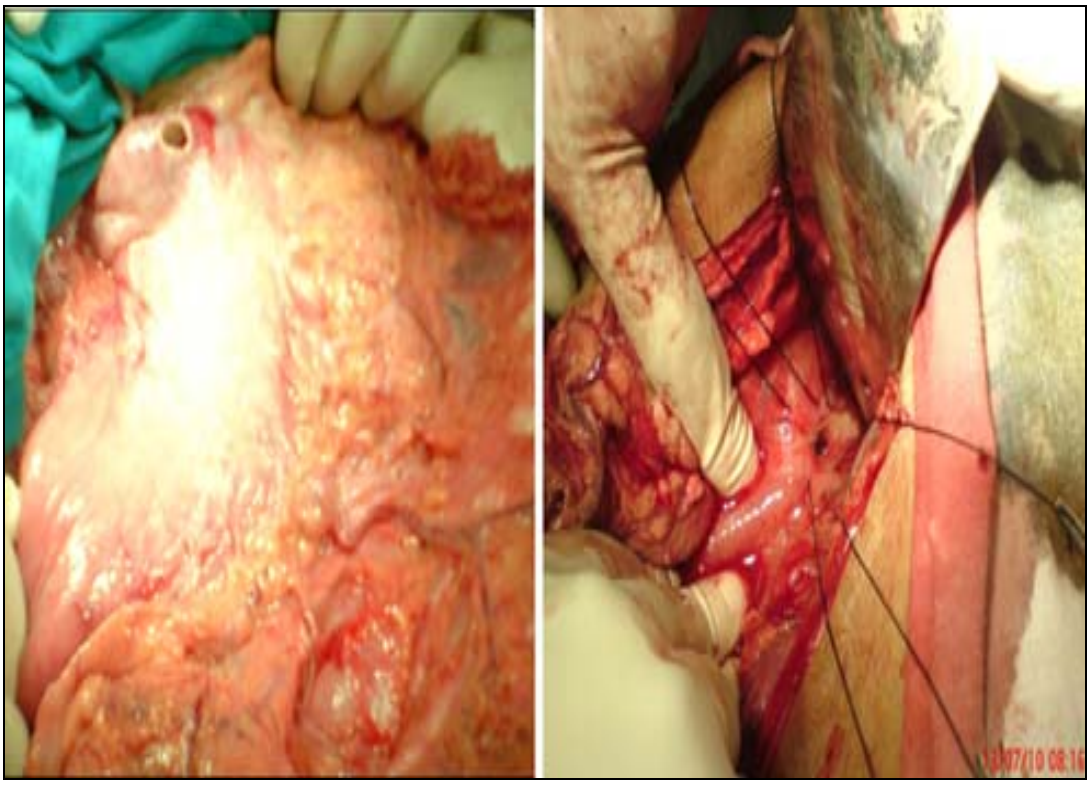
SURGICAL TREATMENT

All cases with perforated ulcer should be operated on within the hour of presentation, if any delay in management >24 hours, increases in mortality and morbidity rates and prolongs hospitalization.

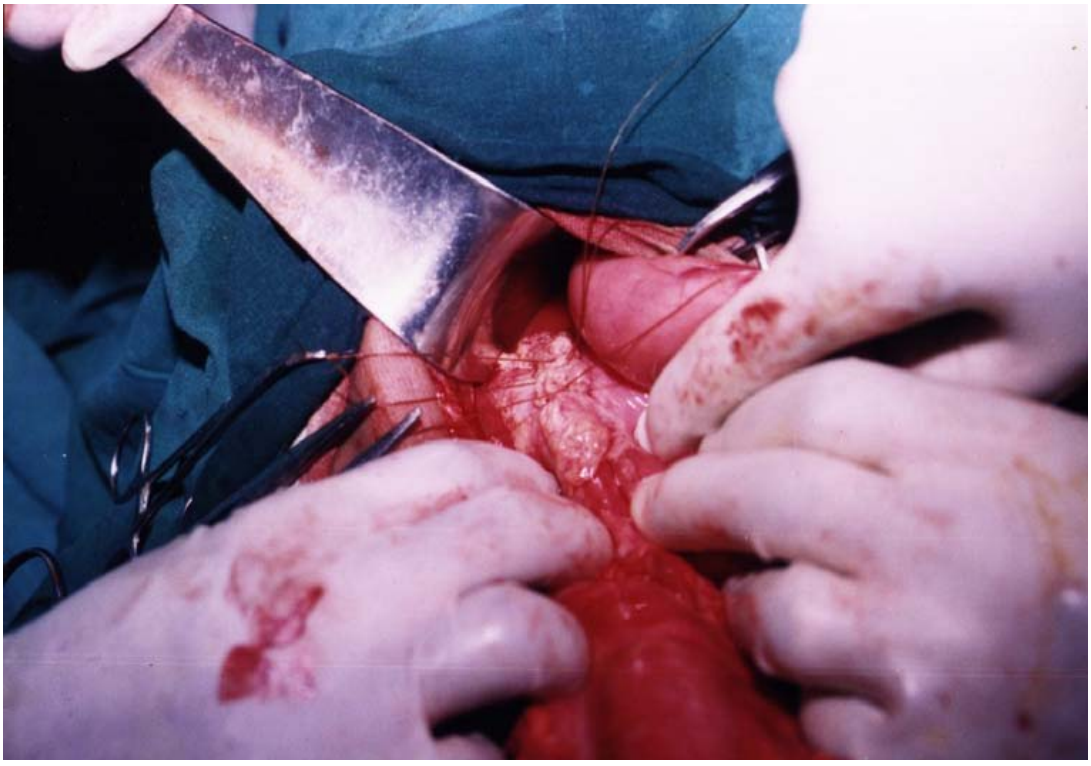
- Simple closure with live omental patch
- Closure with serosal onlay patch
- Distal gastrectomy including ulcer area and reconstruction by billroth I Anastomosis
- Laparoscopic closure of perforated ulcer
- Flank drain and conservative management.

In our hospital RGGGH we commonly do **simple closure with live omental patch.**

SIMPLE CLOSURE WITH ONLAY OMENTAL PATCH



SIMPLE CLOSURE WITH ONLAY OMENTAL PATCH



NON-OPERATIVE MANAGEMENT

Occasionally patients present late that is more than 24 hours after perforation. In this group of patients, non operative management may be considered if, he patient is hemodynamically stable

Generalized peritonitis is absent and Water soluble contrast examination shows no free leak into the peritoneal cavity.

Management of these patients includes nasogastric suction, intravenous proton pump inhibitors, broad spectrum intra venous antibiotics and close clinical observation.

Operation should be immediately considered if clinical deterioration occurs. These patients are prone to development of subphrenic or subhepatic abscess.

This complication usually can be managed with percutaneous catheter drainage. Previous retrospective studies have suggested a more liberal use of non operative management, even in early perforation, as long as no free leak is identified and diffuse peritonitis is absent.

Caution should be exercised in application of this approach to the elderly, frail patient.

Only one third of patients more than 65 years of age were successfully managed non operatively in the hong kong series, compared with nearly 75% of those 40-65 years of age and 100% of those younger than 40% years of age.

Elderly patients are less able to approach, and early operation may be preferable.

Indications for definitive therapy

- Perforation less than 24 hours duration
- Haemodynamically stable, young patients
- No obvious co-morbidity
- Patients with long history of peptic ulcer
- Perforation of an ulcer during anti secretory treatment
- Previous ulcer complication.

Contraindications

Associated medical illness, delay in presentation of more than 24 hours.

POST OPERATIVE MANAGEMENT

- Proton pump inhibitors should be given for 4-6 weeks.
- Antibiotic therapy for H.Pylori should be given.

POST OPERATIVE COMPLICATIONS

- Wound infection
- Intra peritoneal abscess
- Respiratory complications
- Deep vein thrombosis
- Pulmonary embolism

PROGNOSIS

- Prognosis Depends on
- The amount and nature of fluid in the peritoneal cavity
- Larger the perforation poorer the prognosis
- Perforation near the prepyloric region
- Elderly age group
- Associated medical illness
- Longer the time lag

MATERIALS AND METHODS

This study was conducted in RGGGH & Madras medical college, Department of general surgery during the period of August 2012 – December 2012.

The diagnosis of gastric ulcer perforation was that established by the admitting surgeon, clinical features and radiological report and confirmed at operation.

Surgery was defined as urgent (less than 4 hrs between admission and surgery), same day(4-24 hrs) and delayed at a later time during the same hospital admission.

Operative details includes the size,site of perforation and nature of operation performed.

Mortality was defined as death following surgical procedure.

Post operative morbidity was defined in terms of duration of hospital stay and associated complications following surgery.

EXCLUSION CRITERIA

- Acute appendicitis, acute pancreatitis, acute cholecyatitis
- Traumatic gastric perforation
- Accidental gastric perforation during laparotomy
- Cases of delayed presentation with shock and septicemia whose general condition did not warrant any operative management even after all resuscitative measures.

PROFORMA

- Name
- Age/sex
- I.P. No
- Occupation
- Rural/urban
- Socio economic status

- Date of admission
- Date of surgery
- Date of discharge

- History of pain abdomen duration
- History of abdominal distension
- History of acid peptic disease
- History of drug intake
- History of previous surgery
- History of smoking/alcohol
- History of systemic medical illness

EXAMINATION

- Pulse rate
- Blood pressure
- Respiratory rate
- Temperature

PER ABDOMEN

- Guarding
- Rigidity
- Distension
- Liver dullness obliteration
- Bowel sounds

PER RECTAL EXAMINATION

INVESTIGATIONS

- Blood sugar
- Blood urea
- Serum creatinine
- Serum electrolytes
- Complete blood count
- Blood hemoglobin
- Chest X ray
- X ray abdomen erect
- USG abdomen
- ECG

MANAGEMENT

- Time lag
- Operative findings - site
  - Acute/chronic ulcer
  - Peritoneal fluid nature
- Procedure done - simple closure with onlay live omental patch

  Definitive surgery

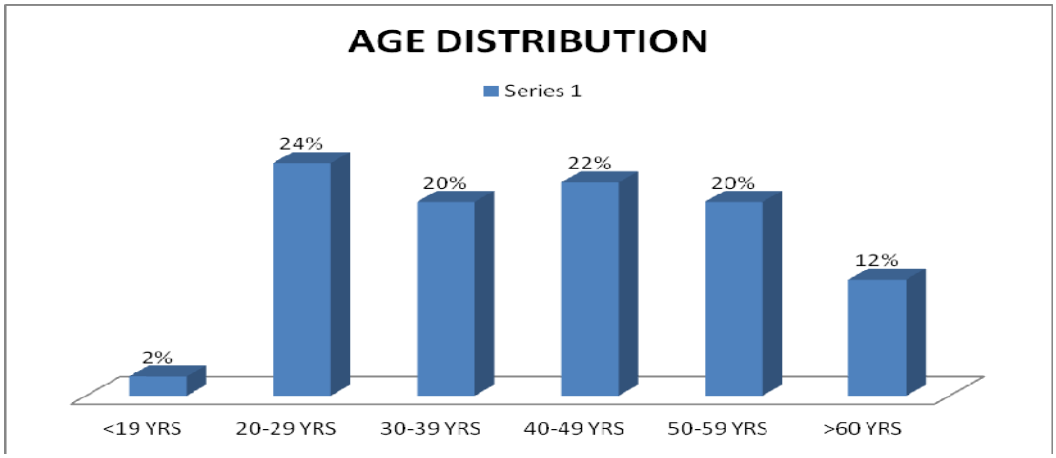
  Non operative treatment
- Post- operative follow up.

RESULTS

- Fifty cases of gastric ulcer perforation were studied.
- All fifty cases underwent laparotomy and the perforation was found in the pyloric antrum.
- Malignant ulcer perforation found in one case.
- All cases were advised to continue anti H.Pylori treatment and proton pump inhibitors for 6 weeks post-operatively.

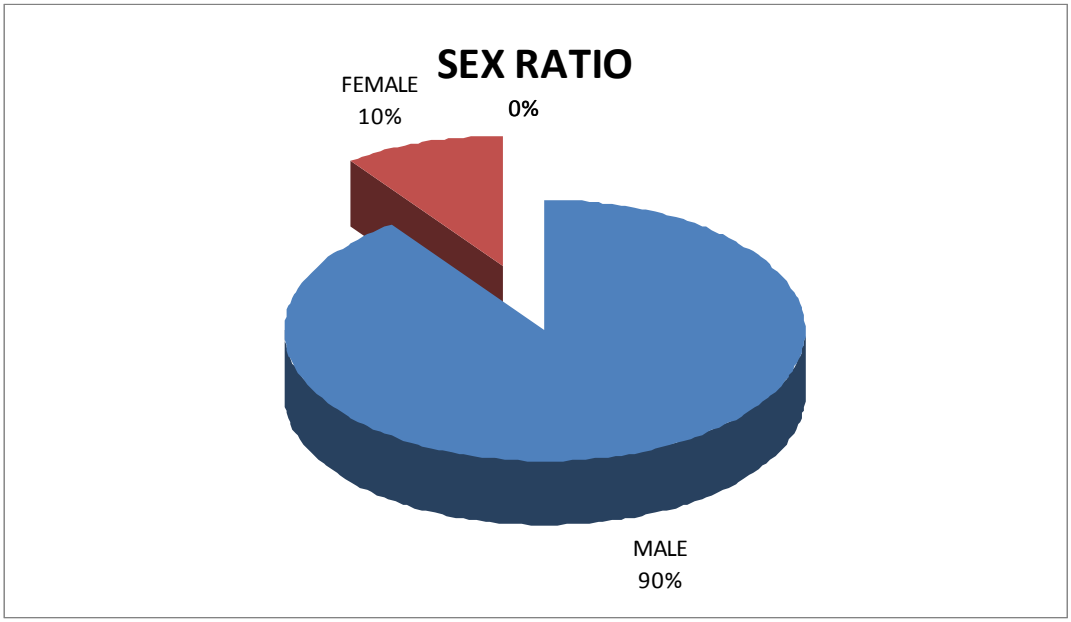
The following observations were made out

AGE DISTRIBUTION



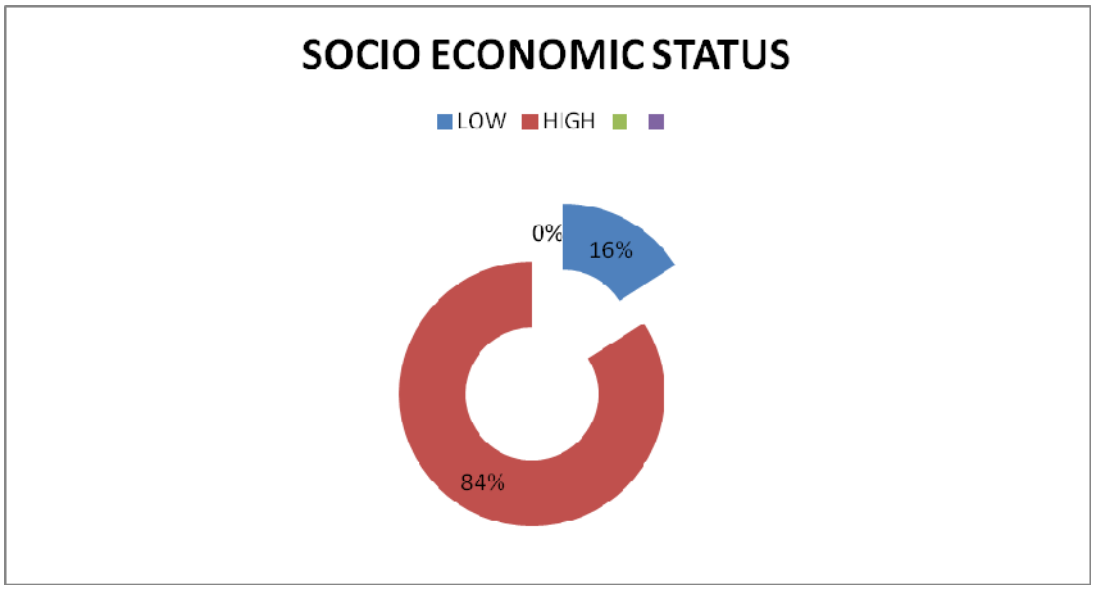
AGE	NO.OF CASES	PERCENTAGE%
<19 YRS	1	2%
20-29YRS	12	24%
30-39YRS	10	20%
40-49 YRS	11	22%
50-59 YRS	10	20%
>60 YRS	6	12%

SEX



SEX	**NO.OF CASES**
Male cases	**45**
Female cases	**5**

SOCIOECONOMIC STATUS



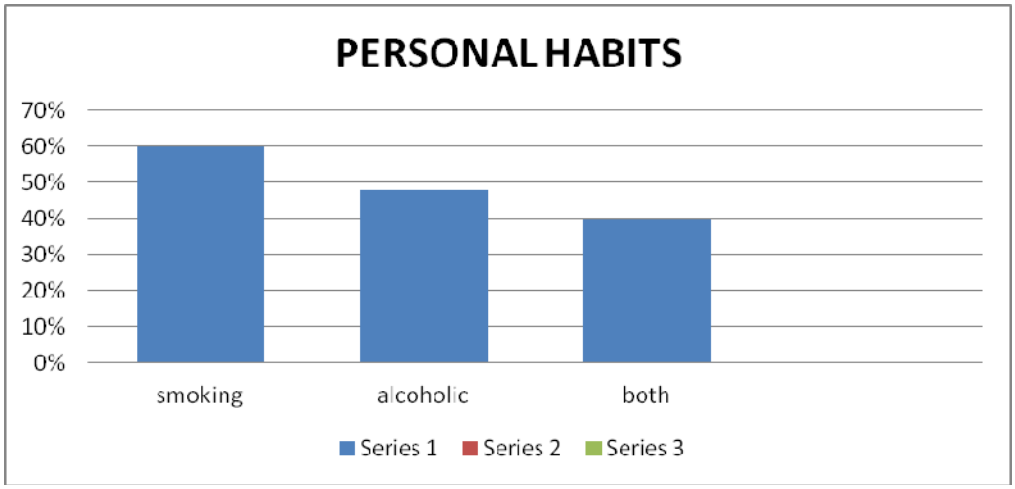
SOCIO ECONOMIC STATUS	NO.OF CASES
Low socio economic status	42
others	8

PERSONAL HABITS

In our study 30 patients are chronic smokers

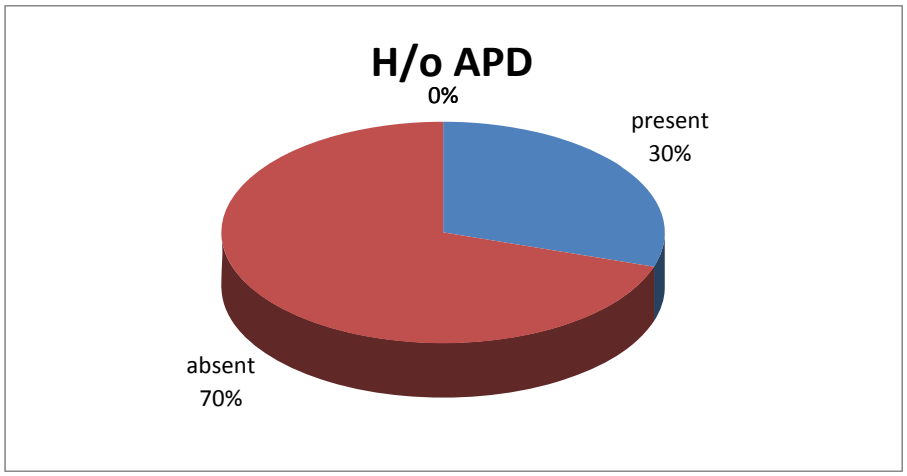
24 patients are chronic alcoholics

20 patients both alcoholic and smokers



HISTORY OF ACID PEPTIC DISEASE

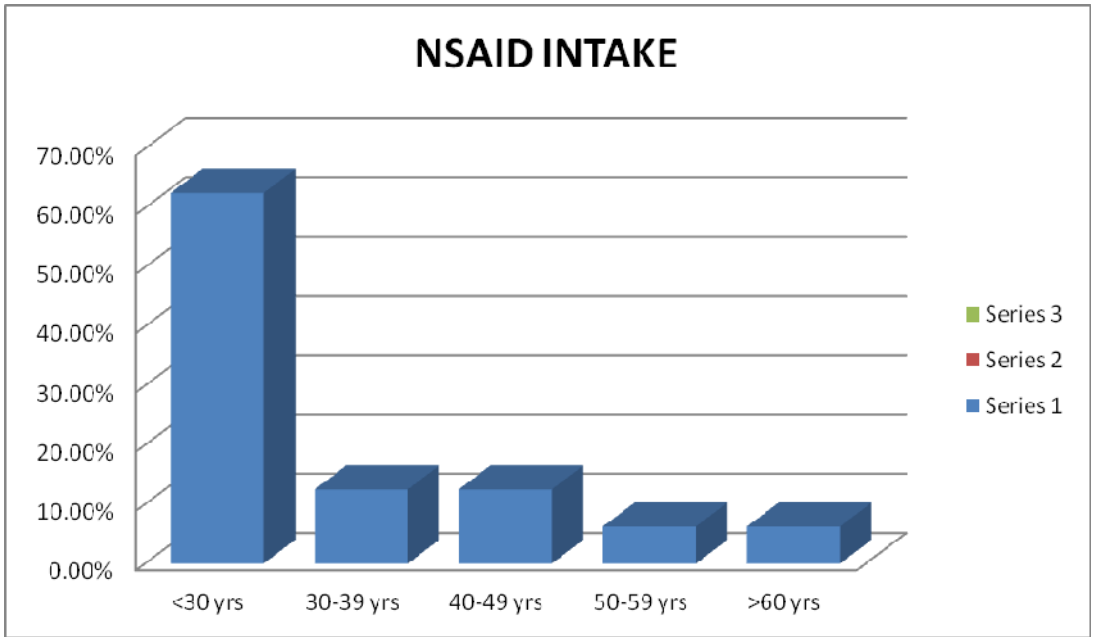
In our study 15 patients gives history of peptic ulcer disease



APD OF APD	NO.OF CASES
History of APD	15 cases
Absent history	35 cases

NSAID INTAKE HISTORY

In our study 16 patients have given history of drug intake before developing gastric ulcer perforation.

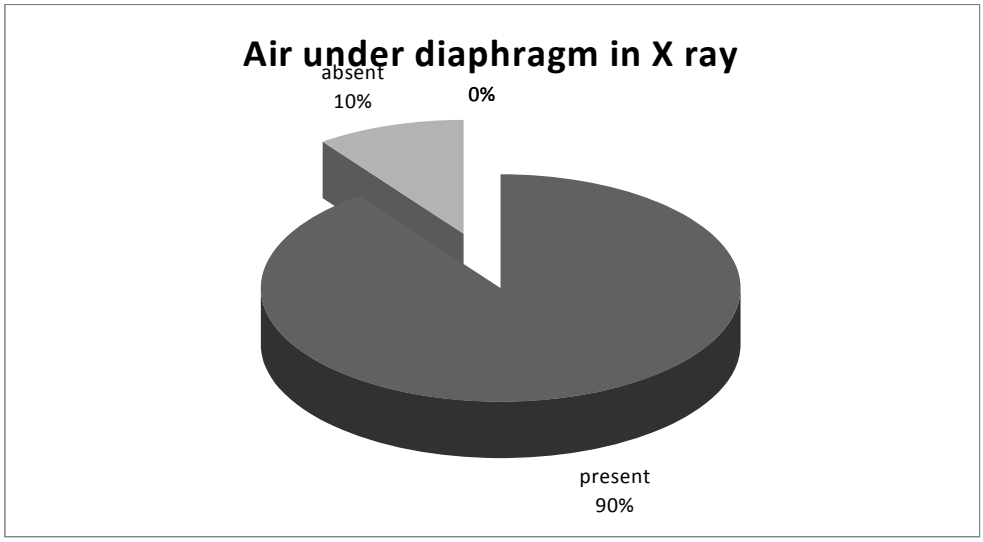


Age	No.of cases	Percentage(%)
<30 years	10	62.5%
30-39 years	2	12.5%
40-49 years	2	12.5%
50-59 years	1	6.25%
>60 years	1	6.25%

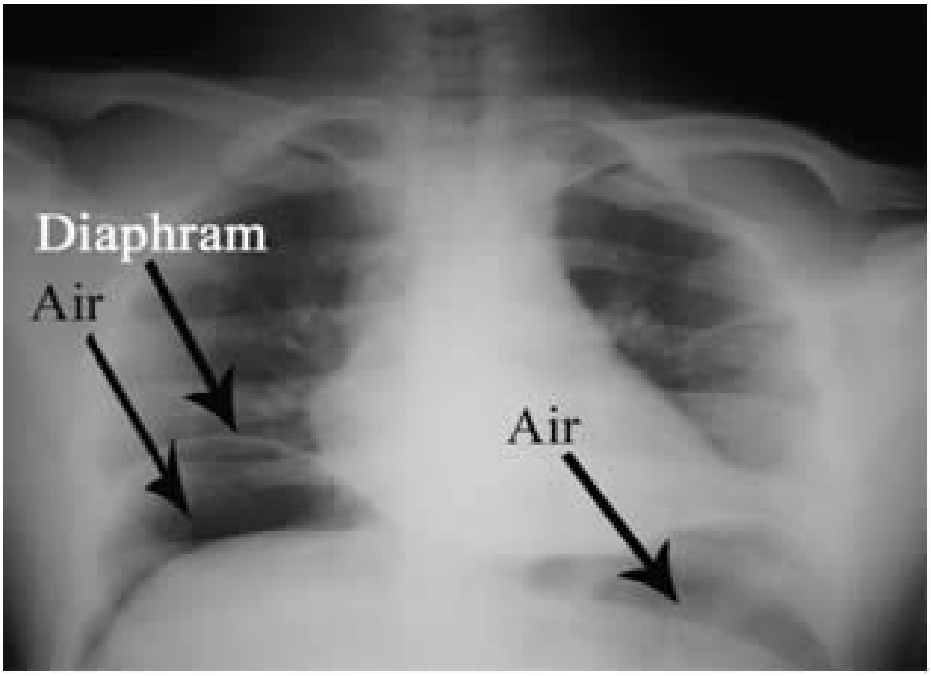
RADIOLOGICAL SIGNS –AIR UNDER DIAPHRAGM

In our study 45 patients X ray erect showed air under diaphragm, CECT was done for those patients X ray negative.

AIR UNDER DIAPHRAGM



CHEST X RAY – AIR UNDER DIAPHRAGM



The presence of free, intra-abdominal gas almost always indicates perforation of a hollow viscus. The commonest cause is perforation of a peptic ulcer.

70% of perforated peptic ulcers will demonstrate free gas, a phenomenon which is almost never seen in case of a perforated appendix or gallbladder.

As little as 1 ml of free gas can be demonstrated radiographically, in either an erect chest or a left lateral decubitus abdominal radiograph, an erect chest film being superior to abdominal radiographs.

Patient should remain in position for 5-10 minutes before the horizontal ray radiograph is taken to ensure that any free gas present has had time to rise to the highest position. In erect radiograph small amount of gas are easily detectable under the right hemidiaphragm, but on left side it may be difficult to distinguish free gas from gastric fundal gas and colonic gas. A left lateral decubitus radiograph will almost always resolve the problem by demonstrating gas between liver and the abdominal wall.

Pneumoperitoneum without peritonitis:

Some patients who present with vague clinical symptoms have unequivocal evidence of pneumoperitoneum in radiographs. However, clinical examination will reveal that there is no evidence of peritonitis or indication for immediate surgery. Some of the causes for such situations

are

i) Silent perforations of a hollow viscus which has sealed by itself (in aged, patient on steroids, unconscious patients).

ii) Post operative pneumoperitoneum

iii) Peritoneal dialysis

iv) Perforated jejunal diverticulosis

v) Perforated cyst in pneumatosis intestinalis

vi) Tracking down from a pneumomediastinum

vii) Stercoral ulceration

viii) Tubal insufflation tests in females

ix) Therapeutic embolisation of an intra-abdominal organ

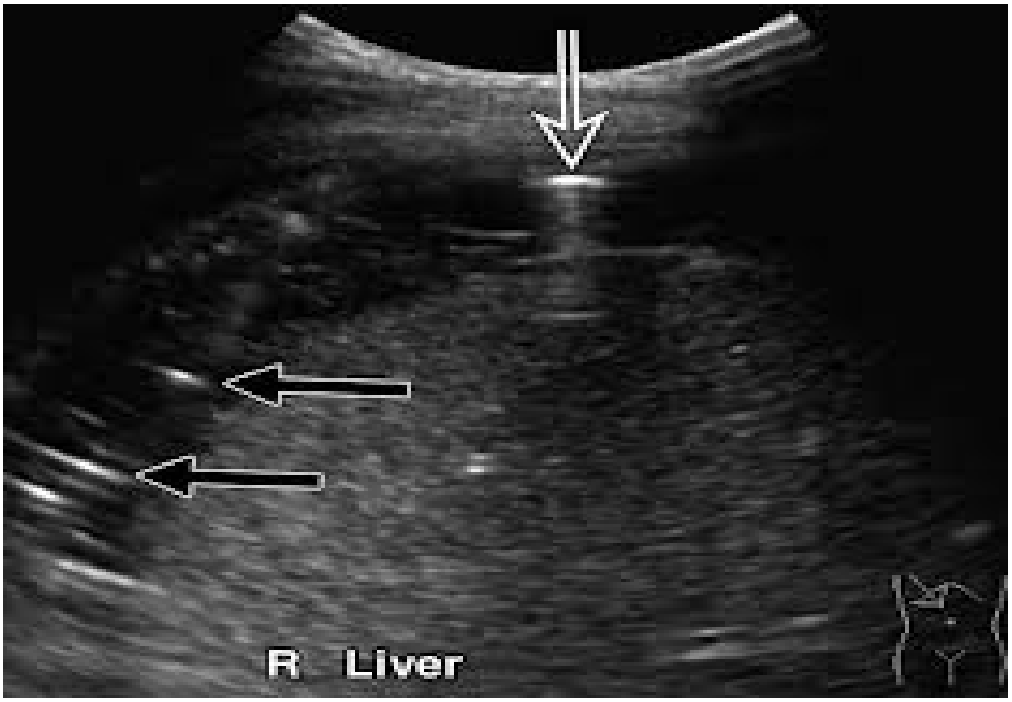
Water-soluble contrast study

Water-soluble contrast (preferably non-ionic) medium about 50ml is given by mouth or injected through a nasogastric tube, with patient lying on his right side.

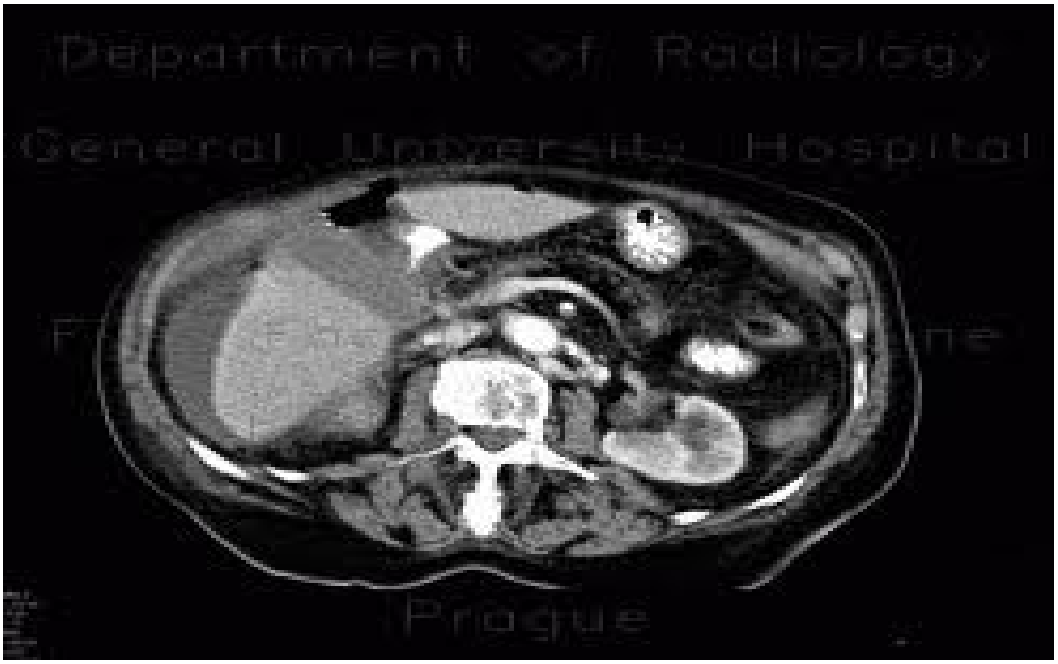
The patient is then examined fluoroscopically or abdominal radiograph repeated after the patient has remained in this position for atleast 5 minutes.

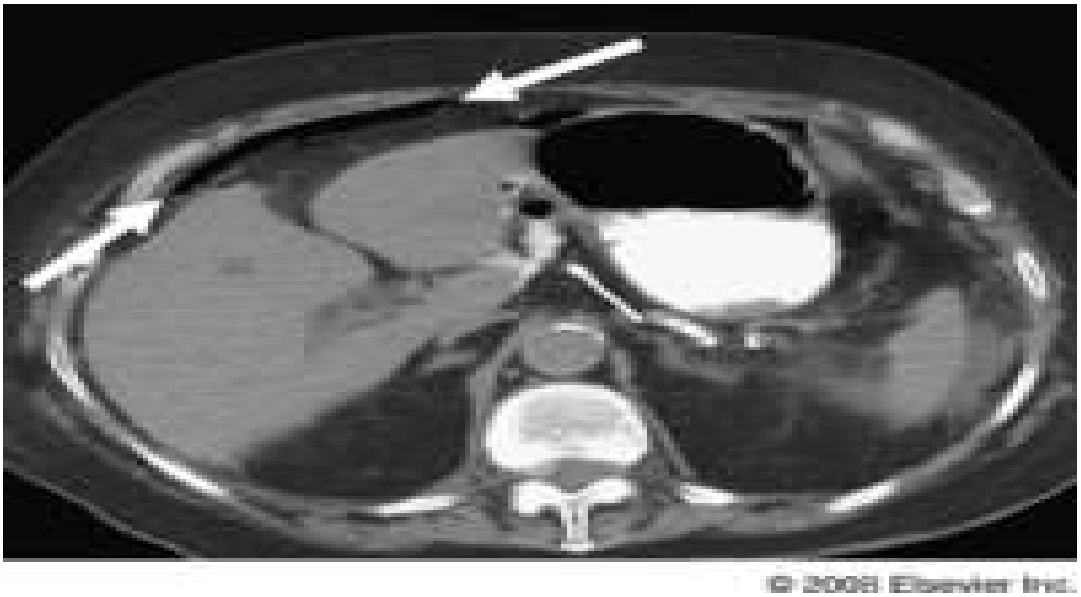
Perforated ulcers will normally demonstrate evidence of a leak of contrastmedium.

USG ABDOMEN



CECT ABDOMEN – PNEUMOPERITONEUM





CT is the most sensitive investigation for detection of free peritoneal gas. Small volumes of free peritoneal gas can be seen over the liver and anteriorly in the mid abdomen.

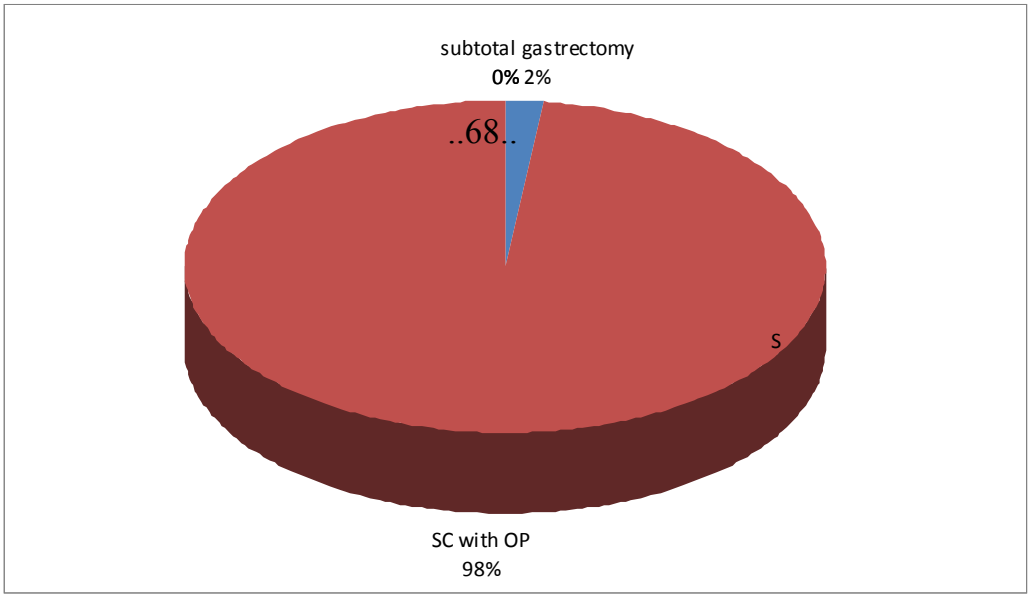
Tiny pockets of free gas can also be seen in the peritoneal recesses. CT is the best investigation to diagnose perforation of posterior wall peptic ulcers, which may be evident by small bubbles of air pocket seen trapped near the wall of stomach or duodenum, near the surface of pancreas or in the mesenteries near the duodenal bulb and stomach.

MANAGEMENT

Out of 50 cases admitted

- 49 cases underwent simple closure with live omental patch.

- 1 case underwent subtotal gastrectomy including ulcer area followed by anterior gastrojejunostomy and jejunojejunostomy.



SYMPTOMS DURATION

In our study the average duration of symptoms was 1.68 days.

TIME DELAY

Time delay defined as from the time of admission to surgery, in our study average delay was 2.22 hours.

PATIENTS HOSPITAL STAY

The average duration of patients hospital stay was found to be 9.36 days.

POST OPERATIVE COMPLICATIONS

- The main post operative complication in our study is wound infection, five case developed wound infection out of fifty cases.
- In these cases wound swab was sent for culture and sensitivity and was treated according to the results.

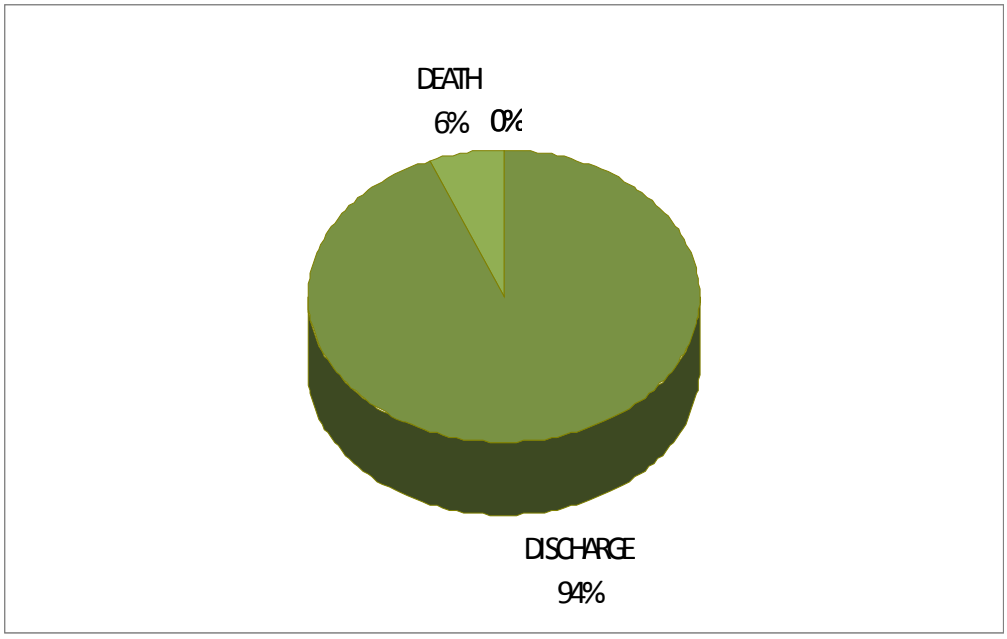
Morbidity Rate – 10%

MORTALITY

Of total 50 cases, three patient expired due to

- septicaemia and
- cardiorespiratory arrest

Mortality rate 6%



MORTALITY

The details of mortality are

S. No	Name	Age/ Sex	Asso Illness	Shock	Symptom Duration	Procedure Done	Death Reason
1.	Dhan bagadur	65/M	DM	+	6 days	SC WITH OP	Septicaemia
2.	Binoy	20/M	-	+	4 days	SC WITH OP	Cardiac arrest
3.	Radha krishnan	62/M	-	+	4 days	SC WITH OP	Septicaemia

POST OPERATIVE MANAGEMENT

- Nil per oral - till 4th POD
- Naso gastric aspiration - till 4th POD
- Pulse, BP chart - upto 48 hrs
- Intake & output chart - till 4th POD
- Intra venous fluids - till 5th POD
- Inj. Pantoprazole 40mg - till 8th POD
- Inj.ceftriaxone 2gm - till 8th POD

Oral fluid were started on 4th POD and semisolid diet started on 5th POD after normal bowel sounds were heard.

DISCUSSION

AGE DISTRIBUTION

- Gastric ulcer perforation is more common in the age group of <30 years in our study.
- 13 cases <30 years - 26%

SEX

- Male to female ratio is 9:1

SOCIO-ECONOMIC STATUS

- 42 patients belong to low socio economic status in our study that is 84%

NSAID INTAKE HISTORY

- 16 patients gives history of drug intake that is 32%

PEPTIC ULCER DISEASE HISTORY

- 15 patients gave history of peptic ulcer disease that is 30%

RADIOLOGICAL SIGNS

- 45 patients X ray showed air under diaphragm, CECT was taken for those patients X ray negative.

SYMPTOMS DURATION

- The average duration of symptoms was found to be 1.68 days.

- The reason being initially patients were treated at various clinics, primary health centres and then referred to our hospital.

TIME DELAY

- The time delay in taking up for emergency laparotomy was 2.22 hours.

- In our hospital all patient with shock initially resuscitated with intravenous fluids and after general condition improved, they were shifted to emergency operation theatre for laparotomy and proceed.

POST OPERATIVE COMPLICATION

- Wound infection was the main post operative complication in our study, particularly those patients with diabetes.

- For these patients wound swab was taken and sent for culture and sensitivity, and treated according to the culture results. This contributed to increase in hospital stay for improvement in general condition.

BIOPSY

Biopsy from the edge of the ulcer in suspected malignant gastric ulcer perforation.

- Total biopsy was taken - 8 cases

- Biopsy positive for malignancy - 1 case

MORTALITY

In our study it depends on

- Age
- Associated comorbid illness like Diabetes etc.
- Time delay from the onset of symptoms and surgery.
- Patients general condition the time of admission (in cases with septicaemia)

All the above are important prognostic factors in gastric ulcer perforation.

Out of these 3 death

- Two patients > 60 years associated with diabetes and delayed presentation
- One patient 20 years with delayed presentation.

CONCLUSION

- Gastric ulcer perforation is now becoming more common in the age group <30 years.
- Associated risk factors including previous history of acid peptic disease and smoking
- NSAIDs intake are associated with increased risk of perforation both younger and elderly patients.
- Wound infection is the common post op complication encounterd in this study.
- Mortality rate is 6% in our study, due to septicaemia and cardiac arrest.
- Males are more affected than females.
- Simple closure with onlay live omental patch closure done in all cases except malignant perforation.
- For malignant perforation we did subtotal gastrectomy including ulcer area along with reconstructive procedure.

Prognostic factors are

- Age
- Comorbid illness
- Duration of symptoms
- Time at presentation
- Patient general condition at time of admission
- Malignant ulcer

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MASTER CHART

S. No	IP No	Name	Age/ Sex	Abd.Pain/ Distension	Duration In Days	Comorbid Illness	H/O Drug Intake	H/O APD	BP At Admission	PR/ Min	X Ray Air	CECT	Delay In Hrs	Procedure Done	Anes	Biop-sy	Malig Nancy	Post OP Co-mp	Hosp Stay Days
1.	71418	shanmugam	45/M	+/-	1	-	-	-	110/70	94	+	-	2	SC with OP	GA	-	-	-	8
2.	73737	Gonidaraj	46/M	+/-	2	-	-	+	90/60	104	+	-	1	SC with OP	GA	-	-	-	10
3.	73784	Manikandan	25/M	+/-	1	-	+	+	100/70	94	+	-	3	SC with OP	GA	-	-	-	8
4.	76023	Mani	55/M	+/+	1	DM	-	-	120/80	88	+	-	2	SC with OP	GA	+	-	-	9
5.	76285	Murugesan	26/M	+/-	1	-	+	-	120/70	92	+	-	3	SC with OP	GA	-	-	-	8
6.	77098	Srinivasan	36/M	+/-	2	-	+	+	90/60	110	+	-	2	SC with OP	GA	-	-	-	10
7.	77552	Raja	30/M	+/-	1	-	-	-	120/70	90	+	-	1	SC with OP	GA	-	-	-	8
8.	79737	Binoy	20/M	+/-	4	-	+	+	80/40	116	+	-	4	SC with OP	GA	-	-	Death 3rd POD	-

S. No	IP No	Name	Age/ Sex	Abd.Pain/ Distension	Duration In Days	Comorbid Illness	H/O Drug Intake	H/O APD	BP At Admission	PR/ Min	X Ray Air	CE CT	Delay In Hrs	Proced Ure Done	Anes	Biop-sy	Malig Nancy	Post OP Co -mp	Hosp Stay Days
9.	79131	Ganapathy	36/M	+/-	2	-	-	-	100/70	108	+	-	2	SC with OP	GA	-	-	-	8
10.	77532	Suresh	33/M	+/-	3	-	+	-	90/70	110	-	+	4	SC with OP	GA	-	-	-	12
11.	82219	Shankar	35/M	+/-	1	-	-	+	110/70	88	+	-	2	SC with OP	GA	-	-	-	8
12.	83281	Kuppan	70/M	+/-	3	HT/DM	-	-	100/70	118	+	-	1	SC with OP	GA	+	-	-	12
13.	83832	Ahamed	40/M	+/-	1	-	-	-	120/80	90	+	-	2	SC with OP	GA	-	-	WI	8
14.	87248	Ganesh	58/M	+/+	4	DM	-	-	80/?	120	-	+	4	SC with OP	GA	-	-	-	13
15.	87395	Tamilselvan	50/M	+/-	1	-	-	-	110/70	88	+	-	3	SC with OP	GA	-	-	WI	9
16.	89281	Pancatsaram	47/M	+/-	1	-	-	-	120/80	94	+	-	2	SC with OP	GA	-	-	-	8
17.	89358	Ramadoss	45/M	+/-	2	HT	-	+	110/60	98	+	-	1	SC with OP	GA	-	-	-	8

S. No	IP No	Name	Age/ Sex	Abd.Pain/ Distension	Duration In Days	Comorbid Illness	H/O Drug Intake	H/O APD	BP At Admission	PR/ Min	X Ray Air	CE CT	Delay In Hrs	Proced Ure Done	Anes	Biop-sy	Malig Nancy	Post OP Co -mp	Hosp Stay Days
18.	89644	Manimaran	26/M	+/-	1	-	+	-	120/80	92	+	-	3	SC with OP	GA	-	-	-	9
19.	93219	Srinivasan	45/M	+/-	5	-	-	-	130/70	96	+	-	2	SC with OP	GA	-	-	-	8
20.	94465	Dhanbagadur	65/M	+/+	1	DM	-	-	70/?	116	+	-	1	SC with OP	GA	+	-	Death 2nd POD	-
21.	96992	Ravanaya	56/M	+/-	1	-	-	+	120/80	94	+	-	3	SC with OP	GA	-	-	-	8
22.	98635	Lakshman	37/M	+/-	1	-	-	-	120/70	98	+	-	2	SC with OP	GA	-	-	-	8
23.	99041	Chellapan	65/M	+/-	2	-	+	+	110/70	112	+	-	3	SC with OP	GA	-	-	-	9
24.	99112	Jagan	22/M	+/-	1	-	+	-	120/80	88	+	-	1	SC with OP	GA	-	-	-	8
25.	99528	Anbu	56/M	+/-	1	HT	-	-	130/90	98	+	-	2	SC with OP	GA	-	-	-	9

S. No	IP No	Name	Age/ Sex	Abd.Pain/ Distension	Duration In Days	Comorbid Illness	H/O Drug Intake	H/O APD	BP At Admission	PR/ Min	X Ray Air	CE CT	Delay In Hrs	Procedure Done	Anes	Biop-sy	Malig Nancy	Post OP Co-mp	Hosp Stay Days
26.	101183	Venkatesan	55/M	+/-	3	-	-	-	100/70	102	+	-	1	SC with OP	GA	-	-	WI	12
27.	101910	Raja	20/M	+/-	1	-	+	-	120/80	94	+	-	3	SC with OP	GA	-	-	-	8
28.	102913	Krisnapillai	50/M	+/+	3	-	-	+	110/70	96	-	+	4	SC with OP	GA	-	-	-	9
29.	102893	Mohana	23/F	+/-	1	-	-	-	120/80	84	+	-	1	SC with OP	GA	-	-	-	8
30.	103638	Raj	75/M	+/-	4	HT	-	-	100/70	102	-	+	4	SC with OP	GA	-	-	-	13
31.	103598	Muthupandy	23/M	+/-	1	-	+	+	110/70	98	+	-	2	SC with OP	GA	-	-	-	8
32.	103911	Periyasamy	45/M	+/-	1	-	-	-	100/70	92	+	-	1	SC with OP	GA	-	-	-	12
33.	105046	Jacob	28/M	+/-	1	-	+	+	120/80	96	+	-	3	SC with OP	GA	-	-	-	8

S. No	IP No	Name	Age/ Sex	Abd.Pain/ Distension	Duration In Days	Comorbid Illness	H/O Drug Intake	H/O APD	BP At Admission	PR/ Min	X Ray Air	CE CT	Delay In Hrs	Procedure Done	Anes	Biop-sy	Malig Nancy	Post OP Co-mp	Hosp Stay Days
34.	105287	Suganya	18/F	+/-	1	-	+	+	120/80	84	+	-	1	SC with OP	GA	-	-	-	10
35.	105453	Sankaran	20/M	+/-	1	-	-	-	110/70	96	+	-	2	SC with OP	GA	-	-	-	8
36.	106210	Radhakrisnan	62/M	+/+	3	-	-	-	80/?	114	+	-	3	SC with OP	GA	+	-	Death 2nd POD	-
37.	106688	Dineshkumar	20/M	+/-	1	-	+	-	110/60	92	+	-	1	SC with OP	GA	-	-	-	8
38.	107988	Sadiqbasha	45/M	+/+	2	HT	+	-	120/70	88	+	-	2	SC with OP	GA	-	-	-	14
39.	108014	Senthilmurugan	37/M	+/-	1	-	-	+	90/60	106	+	-	2	SC with OP	GA	-	-	-	10
40.	108982	vijaya	50/F	+/-	1	DM	-	-	100/70	102	+	-	3	SC with OP	GA	-	-	-	8
41.	106783	Nagarajan	26/M	+/-	2	-	-	+	120/80	106	+	-	2	SC with OP	GA	-	-	-	10
42.	109986	Rajeswari	50/F	+/-	1	-	-	-	100/60	98	+	-	2	SC with OP	GA	-	-	-	8

S. No	IP No	Name	Age/ Sex	Abd.Pain/ Distension	Duration In Days	Comorbid Illness	H/O Drug Intake	H/O APD	BP At Admission	PR/ Min	X Ray Air	CE CT	Delay In Hrs	Procedure Done	Anes	Biop-sy	Malig Nancy	Post OP Co-mp	Hosp Stay Days
43.	110316	Ravikumar	50/M	+/-	1	DM	-	-	110/70	88	+	-	3	SC with OP	GA	-	-	W.I	14
44.	110419	Gnanamurthy	75/M	+/-	1	-	+	-	90/60	104	+	-	2	SC with OP	GA	-	-	-	8
45.	112884	Lakshmi	43/F	+/-	3	-	-	+	90/60	100	-	+	4	SC with OP	GA	-	-	-	10
46.	114294	Murugesan	45/M	+/-	1	HT	-	-	100/70	104	+	-	1	Subtotal gastrectomy	GA	+	+	-	14
47.	114347	Veeramuthu	36/M	+/-	2	-	-	-	110/70	100	+	-	2	SC with OP	GA	-	-	-	8
48.	115350	Samuvel	35/M	+/-	1	DM	-	-	120/80	98	+	-	3	SC with OP	GA	-	-	-	8
49.	116576	Yesuraj	40/M	+/-	1	-	+	-	110/60	94	+	-	2	SC with OP	GA	-	-	-	8
50.	116613	Albert	31/M	+/-	2	-	-	-	110/70	90	+	-	3	SC with OP	GA	-	-	-	10